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     (FILE 'HOME' ENTERED AT 11:04:20 ON 31 DEC 2003)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 11:04:40 ON 31 DEC
     2003
L1         96444 S ADENOVIRUS OR ADENOVIRAL(W)VECTOR
L2         16306 S (DEFECTIVE OR REPLICAT?(3A)DEFICIEN?)(7A)ADENOVIRUS OR ADENOV
L3          5547 S (DEFECTIVE OR REPLICAT?(3A)DEFICIEN?)(7A)(ADENOVIRUS OR ADENO
L4        964643 S EYE OR CORNEAL(W)ENDOTHELIUM OR PHOTORECEPTOR OR BIPOLAR OR G
L5            28 S L3(9A)L4
L6            17 DUP REM L5 (11 DUPLICATES REMOVED)

=> d au ti so ab 1-17 l6

L6   ANSWER 1 OF 17  CAPLUS  COPYRIGHT 2003 ACS on STN
IN   Liu, Lee-Cheng; Newton, Perry; Lai, Shoupeng; Morris, Stephen; Atwell,
     Chad; Hill, Christon; Fitzpatrick, Megan; Cardak, Sami; Lizonova, Alena;
     Qin, Lu; Carrion, Miguel E.; Harris, Brenk K.
TI   Replication-deficient viral vector production methods and compositions
     using complementary animal packaging cells
SO   PCT Int. Appl., 168 pp.
     CODEN: PIXXD2
AB   The present invention provides methods of prepg. viral vector particles
     and viral vector particle compns.  The present invention provides a method
     of producing an adenoviral vector stock by providing a culture of cells
     permissive for growth of adenoviral vectors.  The method is exemplified by
     prepn. of adenoviral vectors contg. E1 and/or E4 deletion and expressing a
     transgene, like TNF, or VEGF, or PEDF, in HEK293 cells which express
     related adenoviral proteins to complement replication defect in adenoviral
     vectors.  Various culture media and culturing conditions are tested to
     improve virus prodn.  Methods for adenoviral particle purifn. through
     microfiber filtration and chromatog. are also disclosed.

L6   ANSWER 2 OF 17  CAPLUS  COPYRIGHT 2003 ACS on STN
IN   Campochiaro, Peter A.
TI   Selective induction of apoptosis to treat ocular disease
SO   U.S. Pat. Appl. Publ., 20 pp.
     CODEN: USXXCO
AB   The invention is directed to a method of prophylactically or
     therapeutically treating choroidal neovascularization, wherein the method
     comprises directly administering to the eye a therapeutic factor or a
     nucleic acid sequence that encodes a therapeutic factor, which is
     expressed to produce the therapeutic factor, to selectively induce
     apoptosis of endothelial cells assocd. with neovascularization of the
     choroid such that choroidal neovascularization is treated prophylactically
     or therapeutically.  The invention also provides a method of
     prophylactically or therapeutically treating ocular neovascularization,
     wherein the method comprises directly administering to the eye a nucleic
     acid sequence encoding a therapeutic factor to promote apoptosis of
     endothelial cells assocd. with neovascularization, such that the nucleic
     acid is expressed thereby producing the therapeutic factor to treat ocular
     neovascularization prophylactically or therapeutically.  Mouse
     eyes were treated with replication-deficient
     adenoviral vectors comprising the coding sequence for
     pigment epithelium-derived factor (PEDF) operably linked to the CMV
     immediate early promoter.  Eyes injected with AdPEDF.10 subretinally or
     intravitreously showed smaller regions of neovascularization, as compared
     to the controls.

L6   ANSWER 3 OF 17      MEDLINE on STN                    DUPLICATE 1
AU   Marmorstein Alan D; Peachey Neal S; Csaky Karl G
TI   In vivo gene transfer as a means to study the physiology and morphogenesis
     of the retinal pigment epithelium in the rat.
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SO Methods (San Diego, Calif.), (2003 Jul) 30 (3) 277-85.
Journal code: 9426302. ISSN: 1046-2023.

AB Our understanding of the morphogenesis of epithelial phenotypes has been greatly advanced by the use of in vitro cell culture systems. However, cell cultures often do not faithfully reconstitute many of the differentiated properties of the cell from which they are derived and cannot be used to examine complex physiologic interactions between adjacent tissues. This is particularly true of the retinal pigment epithelium (RPE). Many plasma membrane proteins, in vivo, exhibit a reversed polarity with respect to other epithelia, and RPE-derived cell lines seldom exhibit these same polarity properties. Furthermore, the interaction between the RPE cell and the neuorsensory retina, or the underlying blood supply, the choroid, is absent in cell culture. Most epithelia are difficult to isolate and study in vivo. The RPE is an exception to this. We have explored several aspects of RPE protein transport properties, vision-related physiology, and disease-related pathophysiology in the eye using in vivo gene transfer and electrophysiologic techniques. By injecting replication-**defective adenoviruses** into the subretinal space of rat **eyes**, we have been able to easily direct the expression of a test protein and follow its sorting and physiologic effects on RPE cells and adjacent tissues. Due to binding and internalization of adenoviral vectors to integrins found on the RPE apical plasma membrane, expression in a healthy eye is essentially confined to the RPE cell, even under control of a cytomegalovirus promotor. The use of varying amounts of adenoviral vector allows for determination of dose-responsive effects and the comparison of multiple mutants of a protein. In addition, there are substantial savings with respect to time and money in comparison to standard transgenic approaches.

L6 ANSWER 4 OF 17 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN

AU Gonzalez, P. [Reprint Author]; Liton, P. B. [Reprint Author]; Caballero, M. [Reprint Author]; Stamer, D. W.; Liu, X. [Reprint Author]; Bodman, M. G. [Reprint Author]; Epstein, D. L. [Reprint Author]

TI SEARCH FOR PROMOTERS TO TARGET GENE EXPRESSION IN SELECTED CELLS OF THE OUTFLOW PATHWAY.

SO ARVO Annual Meeting Abstract Search and Program Planner, (2003) Vol. 2003, pp. Abstract No. 1141. cd-rom.
Meeting Info.: Annual Meeting of the Association for Research in Vision and Ophthalmology. Fort Lauderdale, FL, USA. May 04-08, 2003. Association for Research in Vision and Ophthalmology.

AB Purpose: The purpose of this study is to identify promoters capable of targeting gene expression in different cell types of the outflow pathway. Methods: Perfused anterior segments of human cadaver **eyes** were infected with 107 plaque-forming units of **replication-deficient** recombinant **adenoviruses** expressing the beta-galactosidase reporter gene driven by either the CMV, the VE-Cadherin, or the Matrix GLA promoter. 48 Hours after infection the anterior segments were fixed by perfusion with 1% paraformaldehyde, 0.2% glutaraldehyde, 0.02 % NP40, 0.01 % Na DOC, at 15 mmHg, and stained for beta-galactosidase activity. Paraffin sections of the tissue were analyzed for beta-galactosidase expression. Results: The matrix GLA promoter targets gene expression much more specifically in the TM than the CMV promoter. Expression of this promoter is particularly high in the juxtacanalicular area. The VE-cadherin promoter showed high levels of expression in the aqueous venous plexi and episcleral veins, but did not show any expression in either TM or SC cells. Conclusions: The matrix GLA gene promoter targets HTM cells more specifically than the CMV promoter, while providing high levels of expression. The expression of the VE-cadherin promoter in the aqueous venous plexi and episcleral veins indicates that, at least some viral particles can pass through the HTM and across Schlemm's canal endothelium, and reach the blood stream. Promoters from genes expressed in the cells of the outflow pathway might provide the means for a more specific targeting of gene expression in outflow pathway

cells.

L6 ANSWER 5 OF 17 MEDLINE on STN DUPLICATE 2
AU Vollrath D; Feng W; Duncan J L; Yasumura D; D'Cruz P M; Chappelow A; Matthes M T; Kay M A; LaVail M M
TI Correction of the retinal dystrophy phenotype of the RCS rat by viral gene transfer of Mertk.
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2001 Oct 23) 98 (22) 12584-9.
Journal code: 7505876. ISSN: 0027-8424.
AB The Royal College of Surgeons (RCS) rat is a widely studied animal model of retinal degeneration in which the inability of the retinal pigment epithelium (RPE) to phagocytize shed photoreceptor outer segments leads to a progressive loss of rod and cone photoreceptors. We recently used positional cloning to demonstrate that the gene Mertk likely corresponds to the retinal dystrophy (rdy) locus of the RCS rat. In the present study, we sought to determine whether gene transfer of Mertk to a RCS rat retina would result in correction of the RPE phagocytosis defect and preservation of photoreceptors. We used subretinal injection of a recombinant **replication-deficient adenovirus** encoding rat Mertk to deliver the gene to the **eyes** of young RCS rats. Electrophysiological assessment of animals 30 days after injection revealed an increased sensitivity of treated eyes to low-intensity light. Histologic and ultrastructural assessment demonstrated substantial sparing of photoreceptors, preservation of outer segment structure, and correction of the RPE phagocytosis defect in areas surrounding the injection site. Our results provide definitive evidence that mutation of Mertk underlies the RCS retinal dystrophy phenotype, and that the phenotype can be corrected by treatment of juvenile animals. To our knowledge, this is the first demonstration of complementation of both a functional cellular defect (phagocytosis) and a photoreceptor degeneration by gene transfer to the RPE. These results, together with the recent discovery of MERTK mutations in individuals with retinitis pigmentosa, emphasize the importance of the RCS rat as a model for gene therapy of diseases that arise from RPE dysfunction.

L6 ANSWER 6 OF 17 MEDLINE on STN DUPLICATE 3
AU Kee C; Sohn S; Hwang J M
TI Stromelysin gene transfer into cultured human trabecular cells and rat trabecular meshwork in vivo.
SO INVESTIGATIVE OPHTHALMOLOGY AND VISUAL SCIENCE, (2001 Nov) 42 (12) 2856-60.
Journal code: 7703701. ISSN: 0146-0404.
AB PURPOSE: To determine whether stromelysin gene can be introduced into and expressed in the cultured human trabecular cells as well as in the rat **eye** in vivo through means of a recombinant **replication-deficient adenovirus**. METHODS: Stromelysin cDNA was obtained by reverse transcription-polymerase chain reaction with mRNA extracted from the cultured human trabecular cells after induction with interleukin 1alpha. Adenovirus vector that contains stromelysin cDNA was constructed by cotransfection of pJM17 and pDeltaA.CMV-str into the 293 cells. The expression of stromelysin in the cultured human trabecular cells was assayed by Western blot and zymography. The expression of stromelysin in the trabecular meshwork of the rat eyes was detected by in situ hybridization and immunohistochemistry. RESULTS: The constructed adenovirus vector contained stromelysin cDNA, but no E1 region. Western blot and zymogram revealed that the stromelysin could be expressed and that it possessed enzymatic activity in cultured human trabecular cells. In situ hybridization and immunostaining of the stromelysin showed that the complete form of stromelysin was expressed in the trabecular meshwork, the iris, and the uveoscleral outflow pathway of the rat eye. CONCLUSIONS: Stromelysin, a functional gene, can be transferred in vivo into rat eyes and in vitro into cultured human trabecular cells using a replication-deficient adenovirus vector. This shows the possibility of

gene therapy in glaucoma.

L6 ANSWER 7 OF 17 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
AU Klebe S; Sykes P J; Coster D J; Krishnan R; Williams K A (Reprint)
TI Prolongation of sheep corneal allograft survival by ex vivo transfer of the gene encoding interleukin-10
SO TRANSPLANTATION, (15 MAY 2001) Vol. 71, No. 9, pp. 1214-1220.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0041-1337.
AB Background Modification of a donor cornea by gene therapy ex vivo has potential to modulate irreversible rejection, the major cause of corneal graft failure. Our aim was to transfer the gene encoding mammalian IL-10 to ovine donor corneas and to determine subsequent, orthotopic corneal allograft survival in an outbred sheep model.
Methods. The replicative capacity of ovine **corneal endothelium** was determined by autoradiography after deliberate injury. A replication **defective adenovirus** was used to deliver the lacZ reporter gene to ovine corneas and transfected corneas were organ-cultured in vitro to allow transfection efficiency, duration of reporter gene expression, and toxicity attributable to the vector to be determined. A cDNA encoding full-length ovine IL-10 was cloned into an adenoviral vector that was used to transfect donor corneas ex vivo before transplantation, Orthotopic penetrating corneal transplantation was performed in outbred sheep.
Results. Sheep corneal endothelium was found to be essentially amitotic, Transfection of > 70% corneal endothelial cells was achieved with the viral vector and expression was maintained for 28 days in vitro. IL-10 mRNA was detectable in transfected, organ-cultured corneas for 21 days in vitro. Donor corneas transfected with cDNA encoding IL-10 showed significantly prolonged survival after penetrating keratoplasty (median 55 days, range 19 greater than or equal to 300 days) compared with control corneas (median 20.5 days, range 18-32 days, P=0.011).
Conclusion. Local gene therapy mediated expression of the immunomodulatory cytokine IL-10 has the potential to reduce the incidence of corneal graft rejection and to prolong corneal allograft survival.

L6 ANSWER 8 OF 17 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
AU Glatzel W; Flechsig E; Navarro B; Klein M A; Paterna J C; Bueler H; Aguzzi A (Reprint)
TI Adenoviral and adeno-associated viral transfer of genes to the peripheral nervous system
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (4 JAN 2000) Vol. 97, No. 1, pp. 442-447.
Publisher: NATL ACAD SCIENCES, 2101 CONSTITUTION AVE NW, WASHINGTON, DC 20418.
ISSN: 0027-8424.
AB Targeted expression of foreign genes to the peripheral nervous system is interesting for many applications, including gene therapy of neuromuscular diseases, neuroanatomical studies, and elucidation of mechanisms of axonal flow. Here we describe a microneurosurgical technique for injection of replication-**defective** viral Vectors into dorsal root **ganglia** (DRG). **Adenovirus**- and adenoassociated virus-based vectors with transcriptional competence for DRG neurons led to expression of the gene of interest throughout the first neuron of the sensory system, from the distal portions of the respective sensory nerve to the ipsilateral nucleus gracilis and cuneatus, which contains the synapses to the spinothalamic tracts. Use of Rag-1 ablated mice, which lack all B and T lymphocytes, allowed for sustained expression for periods exceeding 100 days. In immunocompetent mice, long-term (52 days) expression was achieved with similar efficiency by using adeno-associated viral vectors. DRG injection was vastly superior to intraneural injection into the sciatic nerve, which mainly transduced Schwann cells in the Vicinity of the site of inoculation site but only inefficiently transduced

nerve fibers, whereas i.m. injection did not lead to any significant expression of the reporter gene in nerve fibers. The versatile and efficient transduction of genes of interest should enable a wide variety of functional studies of peripheral nervous system pathophysiology.

L6 ANSWER 9 OF 17 MEDLINE on STN DUPLICATE 4
AU Guy J; Qi X; Wang H; Hauswirth W W
TI Adenoviral gene therapy with catalase suppresses experimental optic neuritis.
SO ARCHIVES OF OPHTHALMOLOGY, (1999 Nov) 117 (11) 1533-9.
Journal code: 7706534. ISSN: 0003-9950.
AB OBJECTIVE: To determine if adenoviral-mediated transfer of the gene for catalase (CAT), the reactive oxygen species scavenger, suppresses experimental optic neuritis. CLINICAL RELEVANCE: Gene therapy with CAT delivered by an adeno-associated viral vector was previously shown to suppress experimental optic neuritis. Because the transduction of protein expression with recombinant adeno-associated viral vector is relatively slow, taking weeks to reach full levels, we studied the effects of replication-deficient adenovirus containing CAT in suppressing experimental optic neuritis. Transduction with adenovirus occurs within days of inoculation, thus, it may be more applicable for the treatment of patients with acute optic neuritis. MATERIALS AND METHODS: Replication-deficient adenovirus containing CAT was injected above the right optic nerve heads of SJL/J mice that were simultaneously sensitized for experimental allergic encephalomyelitis. For controls, the left **eyes** were injected with the **replication-deficient adenovirus** without CAT or no virus. The histological effects of CAT on the lesions of experimental allergic encephalomyelitis were measured by computerized analysis of the myelin sheath area (for demyelination), optic disc area (for optic nerve head swelling), the extent of the cellular infiltrate, extravasated serum albumin labeled with immunogold (for disruption of the blood-brain barrier), and the in vivo hydrogen peroxide reaction product. RESULTS: After 1 month, cell-specific catalase activity, evaluated by the quantitation of catalase immunogold, was increased about 2-fold each in endothelia, oligodendroglia, astrocytes, and axons of the CAT-inoculated right optic nerves compared with the control left optic nerves. The increased cellular levels of catalase reduced demyelination by 30%, optic nerve head swelling by 25%, cellular infiltration by 26%, disruption of the blood-brain barrier by 61%, and in vivo levels of hydrogen peroxide by 81%. CONCLUSIONS: Adenoviral-mediated gene transfer increased catalase levels in all optic nerve cell types, and it persisted for 1 month after inoculation. The increased cellular levels of catalase suppressed demyelination and blood-brain barrier disruption at the foci in the optic nerve where prior magnetic resonance imaging and histopathologic studies have demonstrated the demyelinating inflammation of experimental and human optic neuritis. Together, they suggest that gene therapy with CAT may be helpful in the treatment of patients with optic neuritis.

L6 ANSWER 10 OF 17 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
AU Skinner L L (Reprint); Borras T; Epstein D L
TI Effect of **replication deficient adenovirus** vectors on outflow facility of excised pig **eyes**
SO INVESTIGATIVE OPHTHALMOLOGY & VISUAL SCIENCE, (15 MAR 1997) Vol. 38, No. 4, Part 1, pp. 2630-2630.
Publisher: LIPPINCOTT-RAVEN PUBL, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106.
ISSN: 0146-0404.

L6 ANSWER 11 OF 17 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AU Skinner, L. L.; Borras, T.; Epstein, D. L.
TI Effect of **replication deficient adenovirus** vectors on outflow facility of excised pig **eyes**.
SO Investigative Ophthalmology and Visual Science, (1997) Vol. 38, No. 4 PART

1-2, pp. S565.
Meeting Info.: Annual Meeting of the Association for Research in Vision and Ophthalmology, Parts 1-2. Fort Lauderdale, Florida, USA. May 11-16, 1997.
CODEN: IOVSDA. ISSN: 0146-0404.

L6 ANSWER 12 OF 17 CAPLUS COPYRIGHT 2003 ACS on STN
IN Barkats, Martine; Mallet, Jacques; Revah, Frederic
TI Recombinant defective adenoviruses containing glutathione peroxidase DNA and their use disease treatment
SO PCT Int. Appl., 22 pp.
CODEN: PIXXD2
AB The present invention relates to a defective recombinant adenovirus comprising at least a DNA sequence coding for all or an active part of glutathione peroxidase or a deriv. thereof. It also relates to their utilization in therapy and to the corresponding pharmaceutical compns. Recombinant defective adenovirus Ad-bGPx, contg., inserted into the E1 gene, the bovine glutathione peroxidase cDNA controlled by the Rous sarcoma virus LTR, was constructed. 293 Cells infected with this recombinant virus displayed glutathione peroxidase activity.

L6 ANSWER 13 OF 17 CAPLUS COPYRIGHT 2003 ACS on STN
IN Barkats, Martine; Mallet, Jacques; Perricaudet, Michel; Revah, Frederic
TI Defective recombinant adenovirus vectors containing a superoxide dismutase gene and use of the vectors for treatment of neurodegenerative diseases
SO PCT Int. Appl., 25 pp.
CODEN: PIXXD2
AB A defective recombinant adenovirus including at least one DNA sequence coding for all or an active part of a superoxide dismutase or a deriv. thereof. The therapeutic use thereof and corresponding pharmaceutical compns. are also disclosed. Adenovirus Ad-hSOD1, a recombinant defective adenovirus contg. the human SOD1 superoxide dismutase cDNA, was prepd. by homologous recombination of adenovirus Ad-dl1324 and plasmid pLTRIX-hSOD1 in cell line 293. The SOD1 cDNA is fused to the Rous sarcoma virus LTR. Ad-dl1324 has a inactivated E1 region.

L6 ANSWER 14 OF 17 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
AU QI X (Reprint); GUY J; DESHMANE S L; CRYSTAL R G
TI IN-VIVO TRANSFER OF THE HUMAN CATALASE GENE TO THE GUINEA-PIG **EYE** MEDIATED BY A **REPLICATION-DEFICIENT ADENOVIRAL VECTOR**
SO INVESTIGATIVE OPHTHALMOLOGY & VISUAL SCIENCE, (15 FEB 1996) Vol. 37, No. 3, pp. 2969.
ISSN: 0146-0404.

L6 ANSWER 15 OF 17 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AU Qi, X. [Reprint author]; Guy, J. [Reprint author]; Deshmane, S. L.; Crystal, R. G.
TI In vivo transfer of the human catalase gene to the guinea pig **eye** mediated by a **replication-deficient adenoviral vector**.
SO Investigative Ophthalmology and Visual Science, (1996) Vol. 37, No. 3, pp. S640.
Meeting Info.: 1996 Annual Meeting of the Association for Research in Vision and Ophthalmology. Fort Lauderdale, Florida, USA. April 21-26, 1996.
CODEN: IOVSDA. ISSN: 0146-0404.

L6 ANSWER 16 OF 17 CAPLUS COPYRIGHT 2003 ACS on STN
IN Abitbol, Marc; Mallet, Jacques; Perricaudet, Michel; Revah, Frederic; Roustan, Paul; Vigne, Emmanuelle
TI Recombinant defective adenoviruses encoding basic fibroblast growth factors and their use in treatment of neurodegenerative diseases
SO PCT Int. Appl., 27 pp.

CODEN: PIXXD2

AB Recombinant defective adenoviruses comprising a heterologous DNA sequence coding for basic fibroblast growth factor (bFGF), prepn. thereof, and use thereof for treating and/or preventing degenerative neurol. diseases are claimed. Plasmid pLTR IX-hbFGF, contg. cDNA for human basic fibroblast growth factor fused to the LTR of Rous sarcoma virus, was prepd. and used to produce recombinant adenovirus by in vivo homologous recombination with defective adenovirus.

L6 ANSWER 17 OF 17 CAPLUS COPYRIGHT 2003 ACS on STN
IN Briand, Pascale; Perricaudet, Michel
TI **Defective adenoviruses** for use in the gene therapy of **eye** diseases
SO PCT Int. Appl., 20 pp.
CODEN: PIXXD2
AB Defective adenoviruses contg. a foreign gene are described for use in the treatment of eye disease. The adenovirus AdRSV.beta.Gal, an adenovirus 5 carrying a .beta.-galactosidase gene under control of a Rous sarcoma virus promoter was constructed and 107-108 pfu injected into the anterior chambers, vitreous humor, or retrobulbar space of the eyes of C57B1/6 mice. .beta.-Galactosidase activity was found to be widely disseminated in all of the tissues injected.

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L6 ANSWER 16 OF 17 CAPLUS COPYRIGHT 2003 ACS on STN
AN 1995:969572 CAPLUS
DN 124:2557
TI Recombinant defective adenoviruses encoding basic fibroblast growth factors and their use in treatment of neurodegenerative diseases
IN Abitbol, Marc; Mallet, Jacques; Perricaudet, Michel; Revah, Frederic; Roustan, Paul; Vigne, Emmanuelle
PA Rhone-Poulenc Rorer S.A., Fr.
SO PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9526409	A1	19951005	WO 1995-FR374	19950324
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	RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	FR 2718150	A1	19951006	FR 1994-3682	19940329
	FR 2718150	B1	19960426		
	CA 2184755	AA	19951005	CA 1995-2184755	19950324
	AU 9521425	A1	19951017	AU 1995-21425	19950324
	EP 753067	A1	19970115	EP 1995-914419	19950324
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE				
	JP 09510621	T2	19971028	JP 1995-525005	19950324
	ZA 9502563	A	19951221	ZA 1995-2563	19950329
PRAI	FR 1994-3682		19940329		
	WO 1995-FR374		19950324		

L6 ANSWER 17 OF 17 CAPLUS COPYRIGHT 2003 ACS on STN
AN 1995:274955 CAPLUS
DN 122:48488
TI **Defective adenoviruses** for use in the gene therapy of **eye** diseases

IN Briand, Pascale; Perricaudet, Michel
PA Rhone-Poulenc Rorer S.A., Fr.; Institut National de la Sante et de la Recherche Medicale (INSERM)
SO PCT Int. Appl., 20 pp.
CODEN: PIXXD2
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9420146	A1	19940915	WO 1994-FR220	19940228
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	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	FR 2702152	A1	19940909	FR 1993-2438	19930303
	FR 2702152	B1	19950524		
	AU 9461444	A1	19940926	AU 1994-61444	19940228
	AU 693782	B2	19980709		
	EP 687184	A1	19951220	EP 1994-908383	19940228
	EP 687184	B1	20020724		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE				
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	HU 218900	B	20001228		
	JP 08509208	T2	19961001	JP 1994-519650	19940228
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	ES 2181710	T3	20030301	ES 1994-908383	19940228
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	NO 9503329	A	19950824	NO 1995-3329	19950824
	US 2002064870	A1	20020530	US 1998-87156	19980528
	US 2002068052	A1	20020606	US 2001-986797	20011113
	US 2003086907	A1	20030508	US 2002-323876	20021220
PRAI	FR 1993-2438	A	19930303		
	WO 1994-FR220	W	19940228		
	US 1995-513998	A1	19951027		
	US 1998-87156	A1	19980528		
	US 2001-986797	A1	20011113		

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     (FILE 'HOME' ENTERED AT 11:04:20 ON 31 DEC 2003)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 11:04:40 ON 31 DEC
     2003
L1         96444 S ADENOVIRUS OR ADENOVIRAL(W)VECTOR
L2         16306 S (DEFECTIVE OR REPLICAT?(3A)DEFICIEN?)(7A)ADENOVIRUS OR ADENOV
L3          5547 S (DEFECTIVE OR REPLICAT?(3A)DEFICIEN?)(7A)(ADENOVIRUS OR ADENO
L4        964643 S EYE OR CORNEAL(W)ENDOTHELIUM OR PHOTORECEPTOR OR BIPOLAR OR G
L5            28 S L3(9A)L4
L6            17 DUP REM L5 (11 DUPLICATES REMOVED)
L7           199 S L3 AND L4
L8            67 S L3(S)L4
L9            46 DUP REM L8 (21 DUPLICATES REMOVED)

=> d au ti so 1-46 l9

L9   ANSWER 1 OF 46  CAPLUS  COPYRIGHT 2003 ACS on STN
IN   Liu, Lee-Cheng; Newton, Perry; Lai, Shoupeng; Morris, Stephen; Atwell,
     Chad; Hill, Christon; Fitzpatrick, Megan; Cardak, Sami; Lizonova, Alena;
     Qin, Lu; Carrion, Miguel E.; Harris, Brenk K.
TI   Replication-deficient viral vector production methods and compositions
     using complementary animal packaging cells
SO   PCT Int. Appl., 168 pp.
     CODEN: PIXXD2

L9   ANSWER 2 OF 46  CAPLUS  COPYRIGHT 2003 ACS on STN
IN   Campochiaro, Peter A.
TI   Selective induction of apoptosis to treat ocular disease
SO   U.S. Pat. Appl. Publ., 20 pp.
     CODEN: USXXCO

L9   ANSWER 3 OF 46      MEDLINE on STN                    DUPLICATE 1
AU   Marmorstein Alan D; Peachey Neal S; Csaky Karl G
TI   In vivo gene transfer as a means to study the physiology and morphogenesis
     of the retinal pigment epithelium in the rat.
SO   Methods (San Diego, Calif.), (2003 Jul) 30 (3) 277-85.
     Journal code: 9426302. ISSN: 1046-2023.

L9   ANSWER 4 OF 46  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AU   Gonzalez, P. [Reprint Author]; Liton, P. B. [Reprint Author]; Caballero,
     M. [Reprint Author]; Stamer, D. W.; Liu, X. [Reprint Author]; Bodman, M.
     G. [Reprint Author]; Epstein, D. L. [Reprint Author]
TI   SEARCH FOR PROMOTERS TO TARGET GENE EXPRESSION IN SELECTED CELLS OF THE
     OUTFLOW PATHWAY.
SO   ARVO Annual Meeting Abstract Search and Program Planner, (2003) Vol. 2003,
     pp. Abstract No. 1141. cd-rom.
     Meeting Info.: Annual Meeting of the Association for Research in Vision
     and Ophthalmology. Fort Lauderdale, FL, USA. May 04-08, 2003. Association
     for Research in Vision and Ophthalmology.

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IN Kahn, Axel; Mallet, Jacques; Perricaudet, Michel; Peschanski, Marc; Robert, Jean Jacques; Le Gal la Salle, Gildas
PA Rhone-Poulenc Rorer S.A., Fr.; Institut National de la Sante et de la Recherche Medicale (INSERM)
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DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9408026	A1	19940414	WO 1993-EP2519	19930917
	W: AU, CA, FI, HU, JP, NO, NZ, US				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	EP 669987	A1	19950906	EP 1993-920753	19930917
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE				
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L2         16306 S (DEFECTIVE OR REPLICAT?(3A)DEFICIEN?)(7A)ADENOVIRUS OR ADENOV
L3          5547 S (DEFECTIVE OR REPLICAT?(3A)DEFICIEN?)(7A)(ADENOVIRUS OR ADENO
L4        964643 S EYE OR CORNEAL(W)ENDOTHELIUM OR PHOTORECEPTOR OR BIPOLAR OR G
L5            28 S L3(9A)L4
L6            17 DUP REM L5 (11 DUPLICATES REMOVED)
L7           199 S L3 AND L4
L8            67 S L3(S)L4
L9            46 DUP REM L8 (21 DUPLICATES REMOVED)
L10          499 S L1(9A)L4
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L12            0 S L10 AND @PD<19940228
L13            0 S L10 AND @PD<02281994
L14            0 S L10 AND PD<02281994
L15            0 S L10 AND PD<022894
L16           86 S L10 AND PD<19940228
L17           80 DUP REM L16 (6 DUPLICATES REMOVED)
L18          366 S L1(6A)L4
L19           64 S L18 AND PD<19940228

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